IN THE SPECIFICATION:

At page 17, please amend paragraph **[0067]** as follows:

Figure 3 is a graphic representation illustrating that Dam regulates *in vivo* induced genes. [ ]ß-galactosidase expression form S. *typhimurium ivi* fusions in Dam^+ and Dam^- strains grown in LB. The vertical axis shows [ ]ß-galactosidase activities (µ-moles of o-nitrophenol (ONP) formed per minute per A_{600} unit per milliliter of cell suspension x 10^3).

At page 18, please amend paragraph **[0068]** as follows:

Figure 4 is a graphic representation illustrating that Dam represses PhoP activated genes. [ ]ß-galactosidase from *S. typhimurium ivi* fusions grown in minimal medium. The vertical axis shows [ ]ß-galactosidase activities (µ-moles of o-nitrophenol (ONP) formed per minute per A_{600} unit per milliliter of cell suspension x 10^3). The *Dam* genotype is shown below the horizontal axis, and the *phoP* genotypes is shown as black ($PhoP^+$) and gray ($PhoP^-$) boxes.

At page 18, please amend paragraph **[0070]** as follows:

[Figure 6] Figures 6A and 6B are graphs depicting the amount and tissue distribution of *Salmonella* in mice immunized with Dam^- mutants (solid boxes) or not immunized (open boxes) on day 1 and day 5 respectively. PP, Peyer's patches; MLN, mesenteric lymph nodes; CFU, colony forming units.

At page 18, please amend paragraph **[0071]** as follows:

[Figure 7] Figures 7A-7D are graphs depicting amount and tissue distribution of *Salmonella* in mice immunized with Dam^- mutants (solid boxes) or not immunized (open boxes) on days 1, 5, 14 and 28 respectively. PP, Peyer's patches; MLN, mesenteric lymph nodes; CFU, colony forming units.

At pages 67-68, please amend paragraph [00244] as follows:

[0244] All bacterial strains used in this study were derivatives of *S. typhimurium* 14028 (strain **1**). Mutant strains were isogenic to wild type and were obtained or constructed as described (*Dam102*::Mu*d*-Cm and *mutS121*::Tn*10* alleles are in LT2 (strain **7**), a highly attenuated (virtually non-pathogenic) strain as shown in Table 2, were obtained from Dr. John Roth (University of Utah) and Dr. Tom Cebula (The Food and Drug Administration), respectively; these alleles (and additional alleles below) were transduced into virulent strain, 14028, constructing strains **2** and **5**, respectively. *Dam[]Δ232* (strain **3**) was constructed using internal oligonucleotides that serve as PCR primers designed to construct an in-frame 300 bp deletion of defined *Dam* sequence. *dcm1*::Km was constructed according to (Julio, S. M., *et al.*, *Molec. Gen. Genet.*, **258**: 178-181 (1998)); the Km resistance determinant is associated with an internal deletion of >600 bp of *dcm* sequence. The *lrp31*::Km is a null insertion in the *lrp* gene (strain **6**). The Dam overproducing strain (strain **4**) contains *E. coli Dam* on a recombinant plasmid (pTP166) in a wild-type background (Marinus, *et al.*, *Gene*, **28**:123-125 (1984).

At page 70, please amend Table 1 as follows:

TABLE 1

Strain *	Genotype	Oral LD_{50}	I.P. LD_{50} #	Competitive Index (I.P.)[a]
1	"wild type"	$>10^{+5}$	>10	---
2	*dam102::Mud-Cm*	$>10^{+9}$	$>10^{+4}$	$<10^{-4}$
3	*Dam[]Δ232* (non-polar deletion)	$>10^{+9}$	$>10^{+4}$	$<10^{-4}$
4	wild type, (pTP166) (Dam overproducer)	$>10^{+8}$	$>10^{+4}$	$<10^{-4}$
5	*mutS121::Tn10*	10^{+5}	ND	0.9
6	*lrp31::Km*	10^{+5}	ND	10.0
7	LT2	ND	$2 \times 10^{+4}$	ND